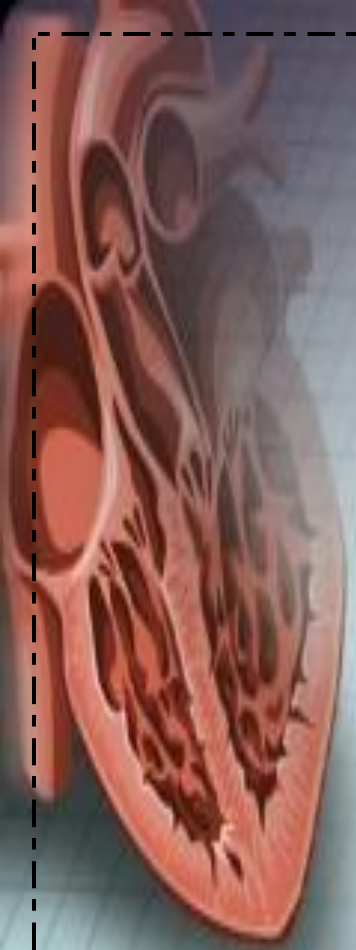

Cardiovascular Block

Physiology Team 430

9th Lecture

Stroke Volume

This Lecture is Done By :

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Stroke Volume:

It is the volume of blood ejected from each ventricle per beat = 70 mls. /beat at rest.

End-systolic volume (ESV):

Is the volume of blood in the left ventricle at the end of contraction, or systole = 50 mls.

End-diastolic volume:

End-diastolic volume (EDV) is the volume of blood in a ventricle at the end of filling (diastole) = 120 mls.

It's determined by 3 variables:

1- End diastolic volume (EDV) = volume of blood in the ventricles at the end of diastole.

2- Total peripheral resistance (TPR) = impedance (resistance) to blood flow in arteries.

3- Contractility = strength of ventricular contraction

The Frank-Starling law of the heart:

It states that, the greater the volume of blood entering the heart during diastole (end-diastolic volume), the stronger the heart contraction and the greater the volume of blood ejected during systolic contraction (stroke volume).

Because greater EDVs cause greater distention of the ventricle, *EDV* is often used with *preload*, which refers to the volume to which the ventricle is stretched before it contract.

An increase in EDV increases the preload on the heart and, through the Frank-Starling mechanism of the heart, increases the amount of blood ejected from the ventricle during systole (stroke volume).

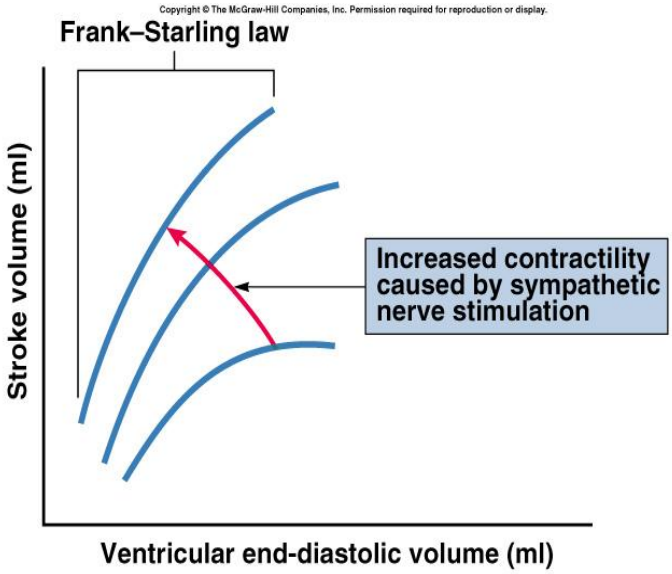
Regulation of Stroke Volume

- EDV is workload (preload) on heart prior to contraction
  - SV is directly proportional to preload & contractility
- Strength of contraction varies directly with EDV
- Total peripheral resistance = afterload which impedes ejection from ventricle
- Ejection fraction is SV/ EDV
  - Normally is 60%; useful clinical diagnostic tool

Frank-Starling Law of the Heart States that:-

Strength of Ventricular contraction varies directly with EDV.

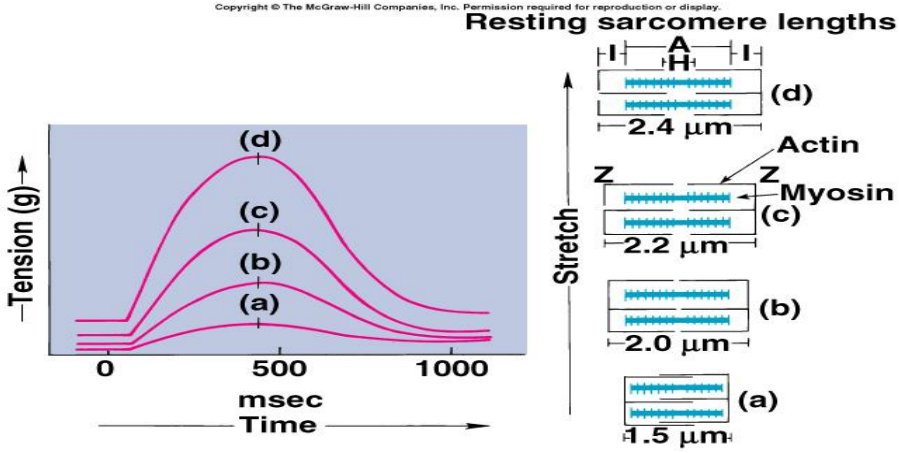
As EDV increases, myocardium is stretched more, causing greater contraction & SV.

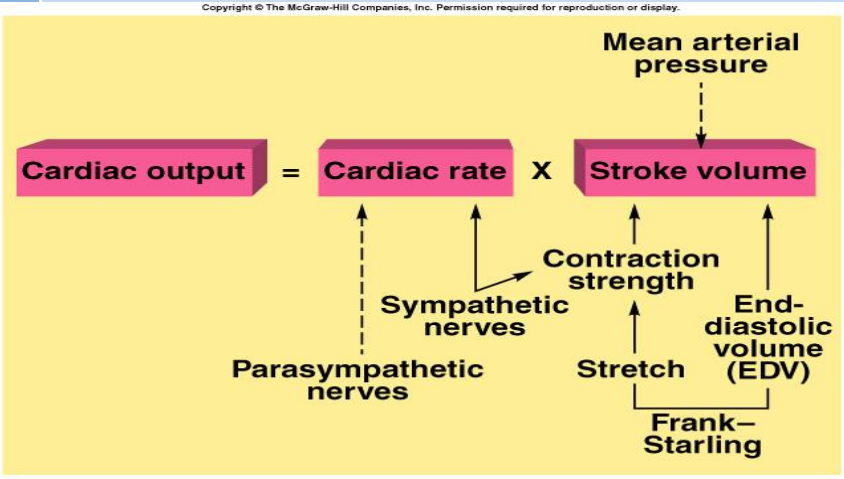


(a) Is state of myocardial sarcomeres just before filling.

Actins overlap, actin-myosin interactions are reduced & contraction would be weak.

In (b, c & d) there is increasing interaction of actin & myosin allowing more force to be developed.





The cardiac output is determined by:

1- preload

2- afterload

* Increased preload increases the stroke volume

* Decreased preload decreases the stroke volume

■ Preload:

It is the **initial stretching of the cardiac myocytes prior to contraction.**

Preload is represented by ventricular end diastolic volume = EDV

When the venous return increases, the end diastolic volume increases (the amount of blood entering the heart and reaches the ventricles increases). Thereby, the muscles of the heart stretch and the stroke volume increase.

■ After Load:

Is the pressure against which the ventricle contract in order to eject blood.

It is increased by increased:-

1- Aortic pressure e.g. Hypertension, and

2- Aortic valve disease e.g. aortic stenosis.

The afterload is closely related to the aortic pressure

When the arterial pressure is reduced, the ventricle ejects blood rapidly which increases the stroke volume and thereby decreases the ESV.

Because of decreased ESV, the ventricles will not fill to the same EDV found before the afterload reduction

Ejection fraction:

Ejection fraction (E_f) is the fraction of the end-diastolic volume that is ejected with each beat.

It is stroke volume (SV) divided by end-diastolic volume (EDV):-

$$E_f = \frac{SV}{EDV} = \frac{EDV - ESV}{EDV}$$

1-intrinsic regulation: in response to changes in the volume of the blood flowing into the heart.

2-extrinsic regulation: control of heart rate and strength of heart pumping by the autonomic nervous system.

Example:

1. Nervous:

*** Sympathetic: increase HR & SV.**

*** Parasympathetic: decrease HR.**

2. Chemical:

*** Potassium**

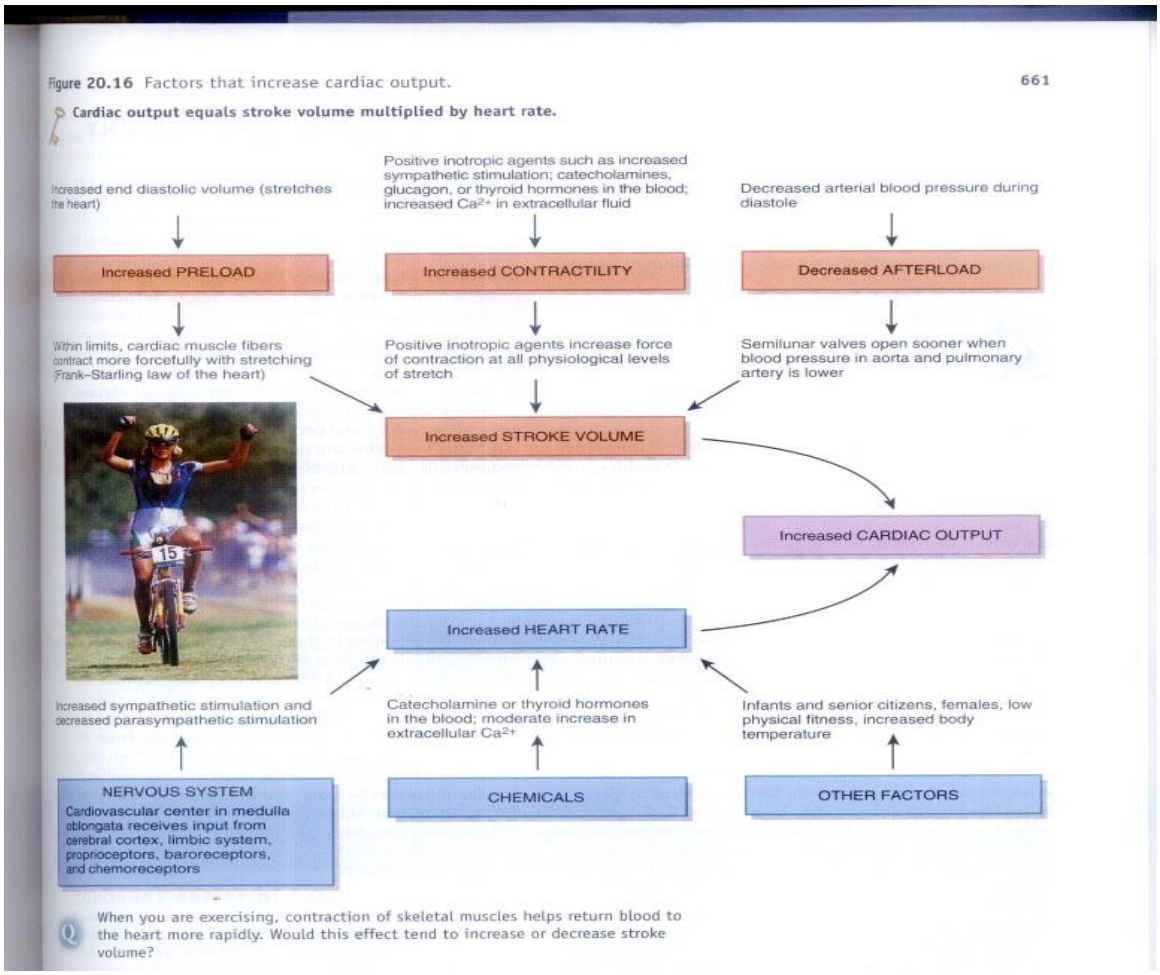
*** Calcium.**

*** Thyroxin.**

*** Catecholamine**

Further reading (just to understand more): من هنا إلى آخر شيء كله فقط للقراءة لتفهم الموضوع كله

Just Read:



Starling law:

when the venous return increases the EDV increases and then the preload increases which is the initial stretching of the cardiac myocytes prior to contraction. The myocyte stretching increases the sarcomere length, which cause an increase in the troponin C calcium sensitivity and cause an increase in the rate of cross bridge attachement and detachment and the amount of tension developed by the muscle fiber. And that causes an increase in the force generation.

This mechanism enables the heart to eject the additional venous return, thereby increasing stroke volume.

Frank-Starling law (intrinsic regulation of CO):

1- The ability of the heart to change its force of contraction and therefore stroke volume in response to change in venous return

2- The ability of the heart to pump all the blood coming to it without allowing systemic stasis, within limits.

Factors affecting EDV:

Increase:

Stronger atrial contraction.
Increased total blood volume
Increased venous tone.
Increased sk m pump.
Increased negative intrathoracic pressure.

Decrease:

Standing
Increased intrapericardial pressure.
Decreased ventricular compliance

Factors affecting afterload:

Vascular tone (and therefore blood pressure)
Aortic stiffness
Myocardial tension (affected by hypoxia, volume overload)
Metabolic rate
Preload
Valvular regurgitation